Armed Forces College of Medicine

AFCM

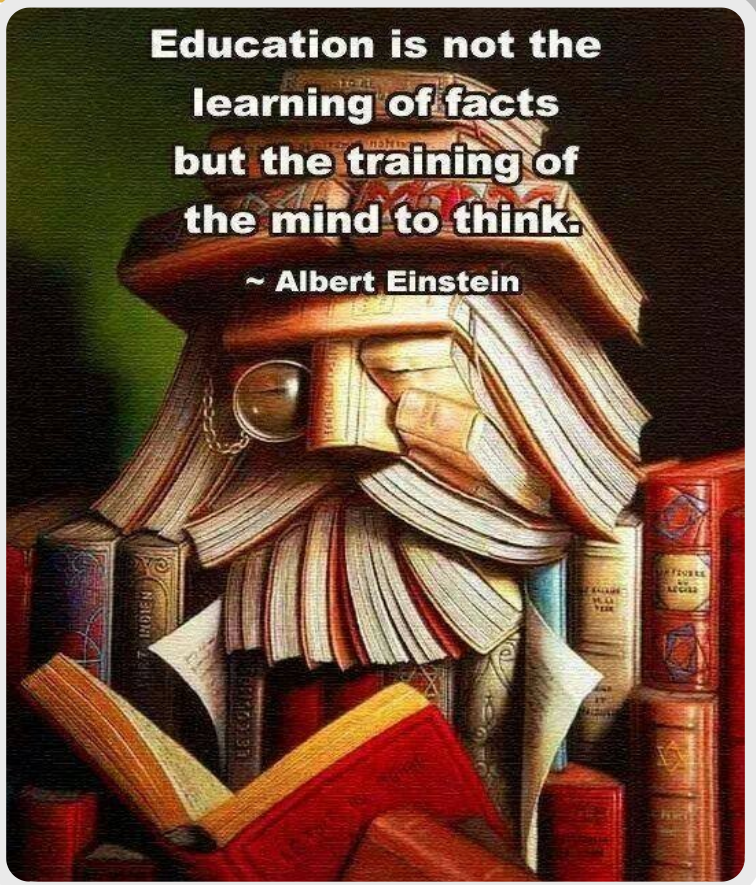
Cardiopulmonary module

Clinical Integrated Cases

Session 5

Basic Science Principles for Clinical Reasoning

1- Critical thinking
2- Introduction to common forms of disease
3- Apply basic knowledge in a realistic clinical scenario.
4- Interpersonal skills-(share and apply your knowledge)… communication…patient safety
5- Life long self-directed learning



Case co-ordinators

Prof. Drs. Manal Hassan, Tamer Elwasify, Lamiaa, Bassant

Case

Easy fatigue with dyspnea on lying in bed

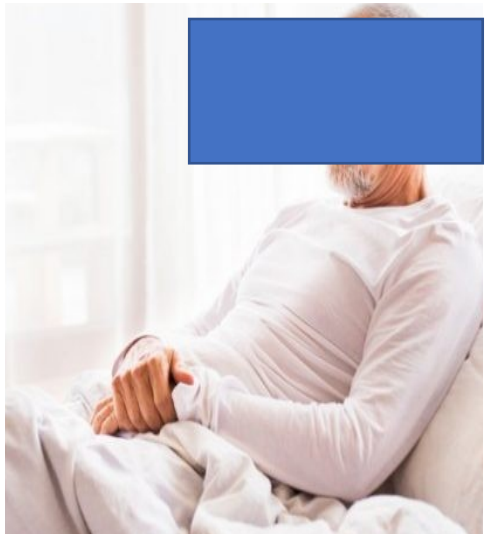
Easily fatigue with dyspnea on lying in bed

Mr Medhat is a 61 year old man who works as an information system consultant. Three months ago, he began to notice **increased fatigue** with **mild shortness of breath**.

Mr Medhat said that he used to work full days. He used to ride his bike for few Km and climb 4 flights of stairs at work without any difficulty. Three weeks ago, he could climb only one flight of stairs before he gets shortness of breath. He stopped riding his bike and gets to sleep early.

Easily fatigue with dyspnea on lying in bed

Medhat started to **wake up at night with episodes of shortness of breath requiring him to sit up**. Medhat had **cough** with **white frothy sputum**.



Two days ago, Medhat found that it was **impossible to lie flat on his back in bed and started sleeping in a chair**.

He thought he might have pneumonia because he was so short of breath

Past history: hypertensive for 20 years. 5 years ago he experienced an **attack of chest pain that was on and off for one week for which he received drugs for gastritis.**

Family history: His father had coronary artery disease and heart failure but died at age of 89.

Easily fatigue with dyspnea on lying in bed

General examination

Vital signs:

Pulse: **112/min regular with occasional dropped beats**

Blood pressure: **140/90 mmHg**

Respiratory rate: **23 cycles/min**

Temperature: **37ºC**

No cyanosis

Trace of jaundice

Neck vein examination:

Elevated, Congested ar
pulsating

Easily fatigue with dyspnea on lying in bed

Cardiac examination:

Apical impulse was **shifted laterally** and downward

Third heart sound was heard…**gallop**

Chest examination:

Bilateral basal crepitations

Abdominal examination:

Tender hepatomegaly with ascites

Leg examination:

Bilateral lower limb

edema

1- List the patient problems

What is your provisional diagnosis

NB. Causes of LL edema
-Cardiac edema
-Hepatic edema
-Renal edema
-Nutritional edema
-Orthostatic edema

Generalized edema with systemic venous congestive symptoms, most probably due to biventricular heart failure

Medhat found that it was impossible to lie down in bed and started sleeping in a chair (orthopnea).

2- Explain the orthopnea complain of Medhat……..

Mechanism of orthopnea

Congested Pulmonary Circulation

- **Failing heart is unable to cope with the extra volume of blood reaching it on lying flat**

↓

Increased pulmonary venous & capillary hydrostatic pressure

↓

Pulmonary interstitial edema

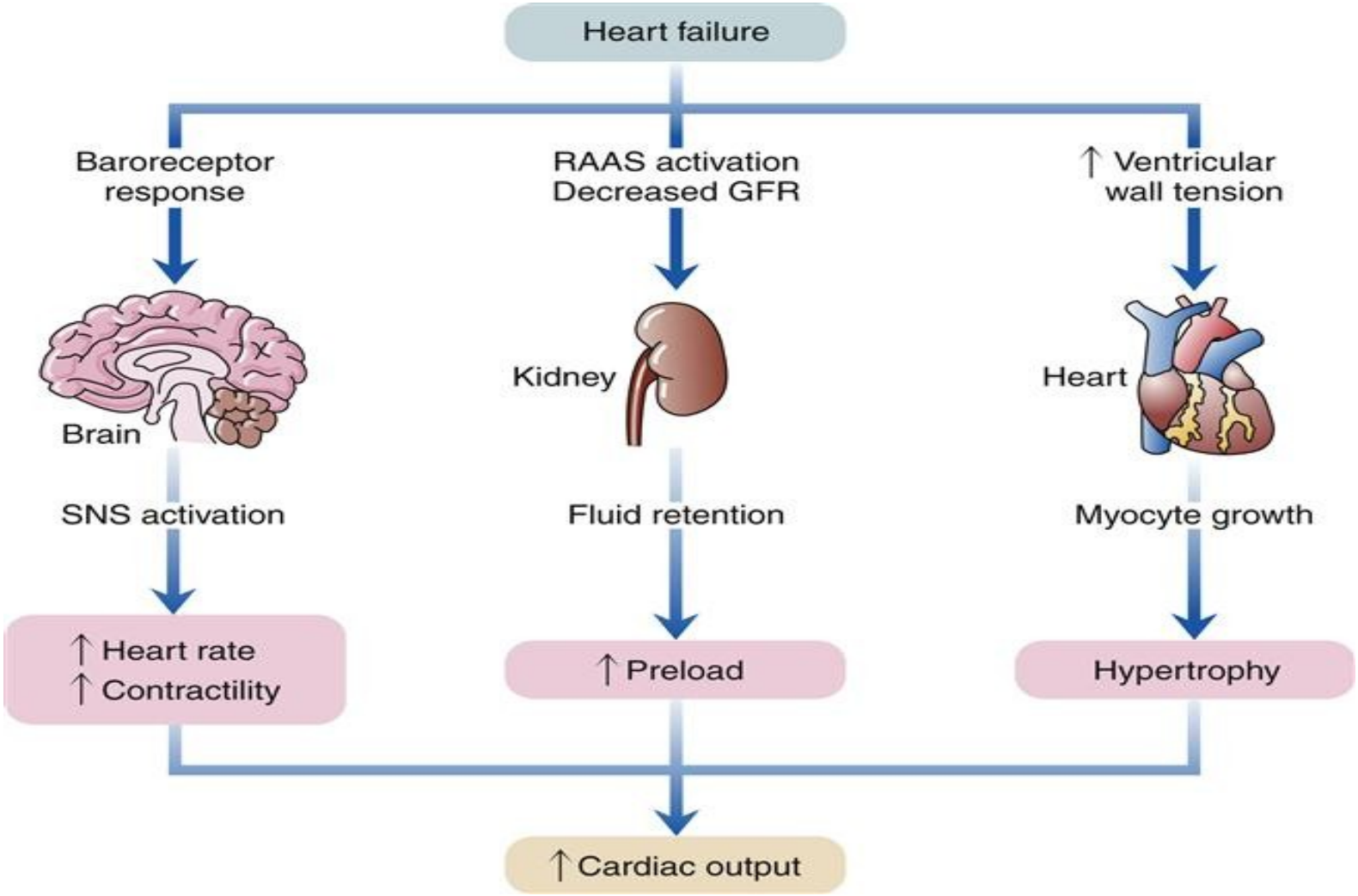
Limited Diaphragmatic Descent (Ascites)

→ Decreased vital capacity of the lung

Pulse: 112/min

Explain the rapid pulse rate in Medhat

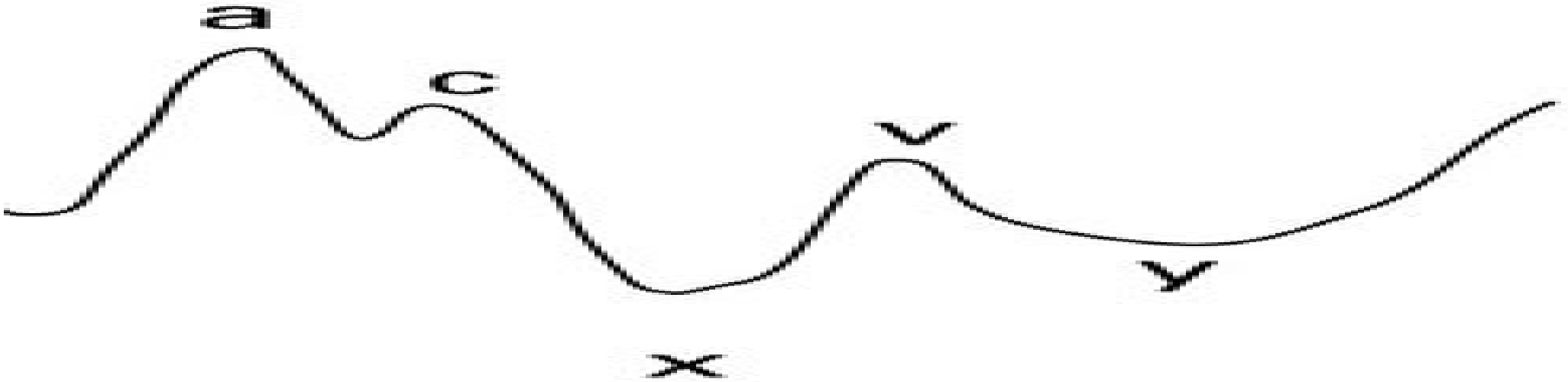
Increase sympathetic activity → increase heart rate (compensatory effect)



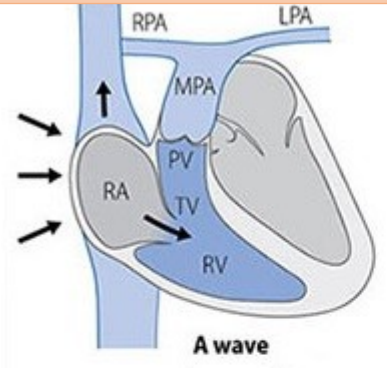
https://basicmedicalkey.com/wp-content/uploads/2016/09/F00019Xf19-02-9781455726509.jpg

Neck vein examination:
Elevated, Congested and pulsating

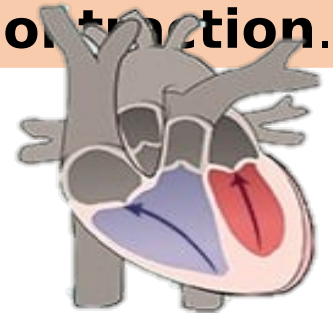
3- What are venous pulsations that could be seen in the neck?



a wave
Ascending limb → **Atrial systole**
Descending limb → Blood flowing into the right ventricle

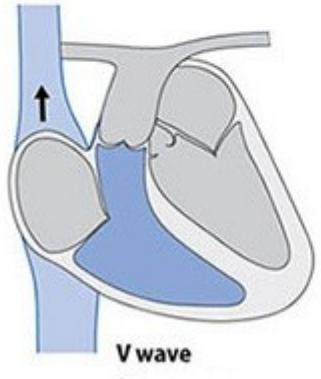


c wave is due to upward bulge of the tricuspid valve during **isometric contraction**.

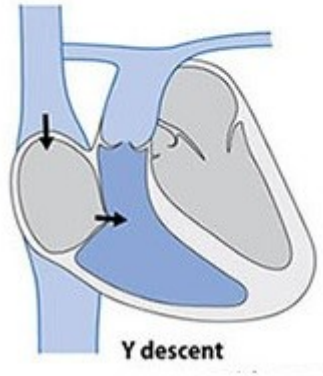


x descent
is due to downward bulge of the tricuspid valve during **maximum ejection**

v wave is due to accumulating **VR in right atrium**



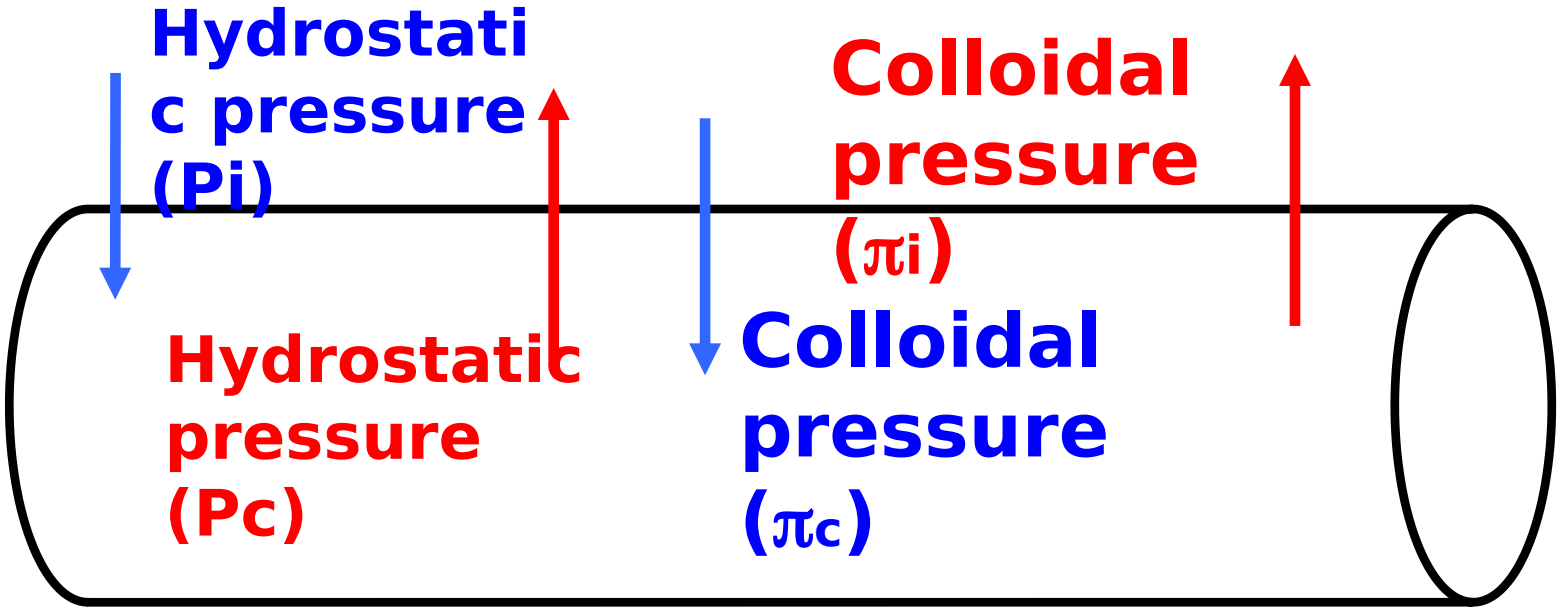
Y descent
is due to **ventricular rapid filling**



Leg examination:
Mild bilateral lower limb edema

4- Explain the lower limb edema in Medhat

Factors affecting the tissue fluid formation



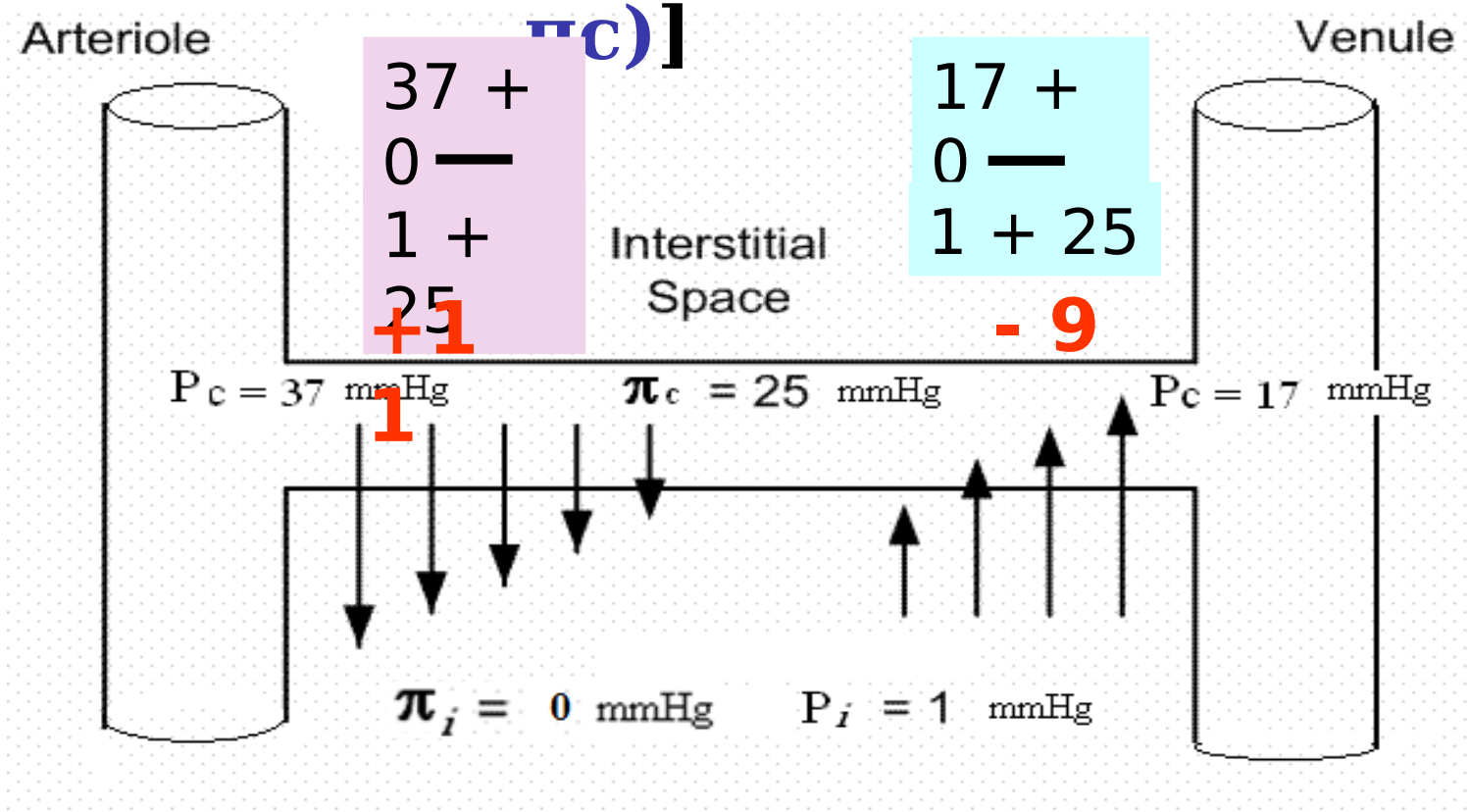
Net force = [(Pc + πi)] – [(Pi + πc)]

Filtration ∝ Net force [(Pc + πi)] – [(Pi + πc)]

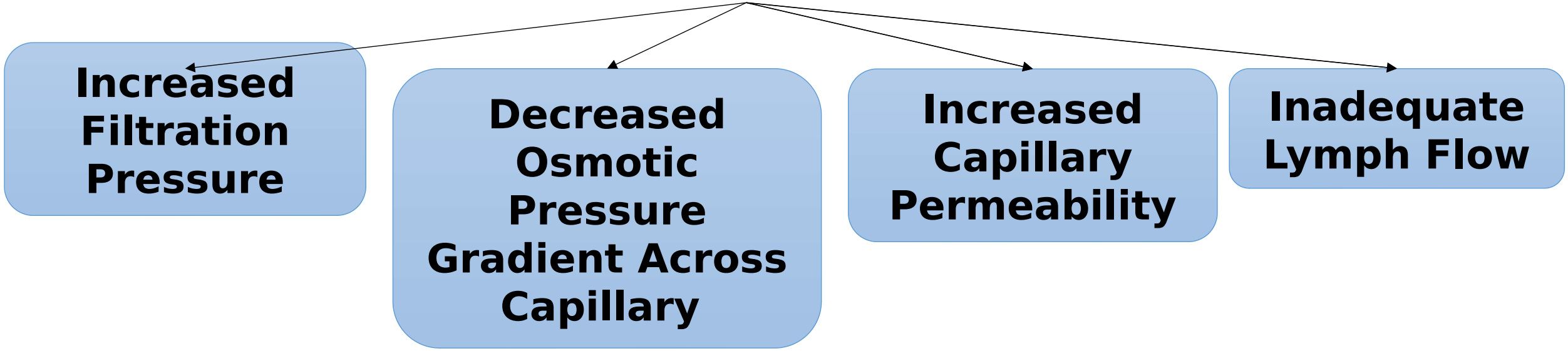
Filtration = K [(Pc + πi)] – [(Pi + πc)]

Starling forces

Net force = [(Pc + πi)] - [(Pi + πc)]



Edema



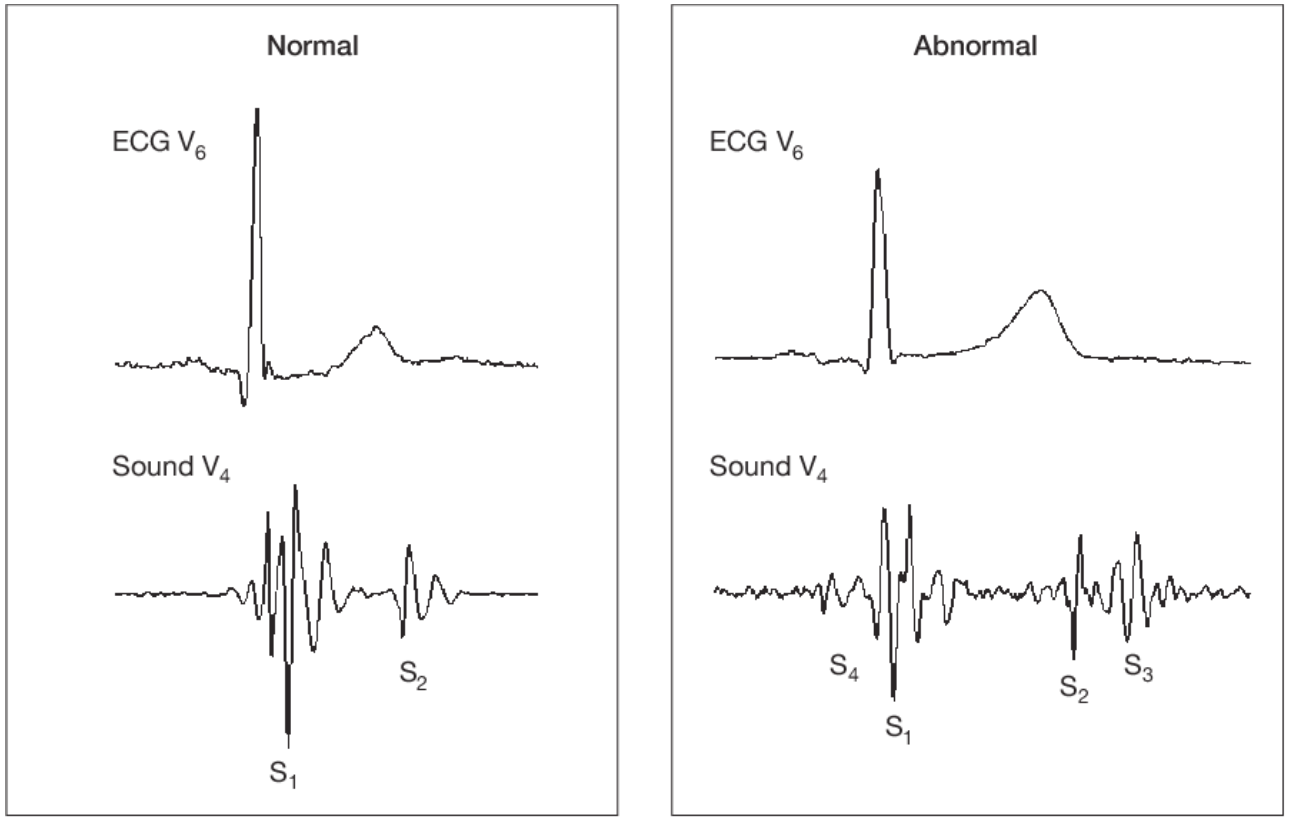
Medhat has heart failure → Increased venous pressure → increased filtration pressure

Third heart sound was heard…gallop

What is the cause of the third heart sound heard in this patient? What's its timing?

Third heart sound (S3)

- It's the sound of ***gush of blood into the ventricle***, causing vibration of the relaxed ventricular wall during ***rapid filling phase*** (diastole).
- This is normally non-audible.
- It indicates a "lax" ventricular wall, eg: in dilated cardiomyopathy, heart failure, ……..



Representative phonocardiographic recordings from the V_4 position and electrocardiographic (ECG) record-

https://d3i71xaburhd42.cloudfront.net/4771d2210bf442b0bb2d7b57881bdb196f0086fe/2re1-1.png

5- What are the investigations to be done to Medhat?

- **Echocardiography**
- **ECG**
- **Chest x-ray**

- **labs (CBC , eGFR ,LFTs ,Thyroid functions , natriuretic peptides ,ferritin , TIBC)**

- **Coronary angiography**
- **Right side heart catheterization and coronary angiography**
- **Genetic testing**
- **cardiac MRI**

Welcome back

Easily fatigue with dyspnea on lying dow

The doctor asked for the following **investigations**

- **ECG**
- **Echocardiography**
- **Chest x-ray**

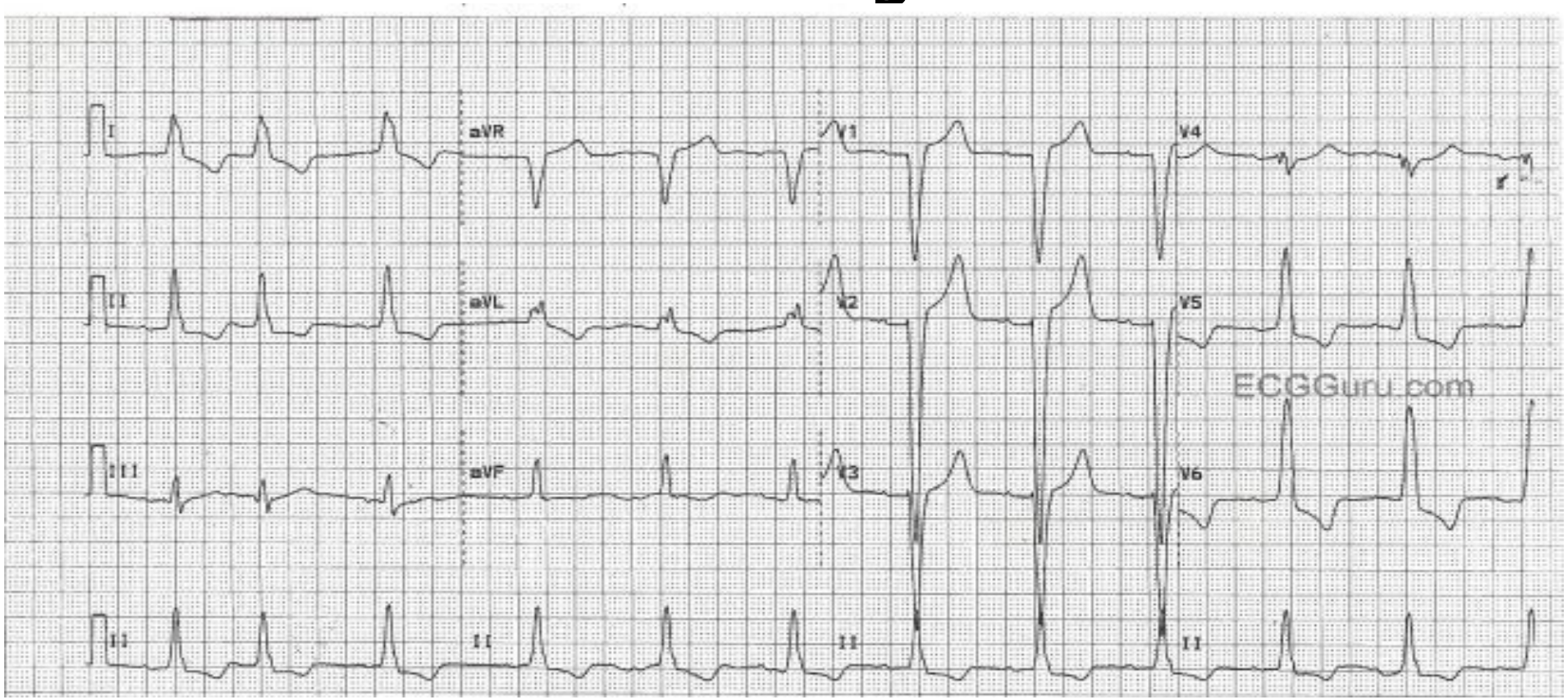
Blood sample:

- CBC, urea and creatinine
- Liver function tests
- Natriuretic peptide biomarkers: **BNP and aminoterminal pro B type natriuretic peptide**
- Serum sodium and potassium

Easily fatigue with dyspnea on lying dow

Results of Medhat investigation:

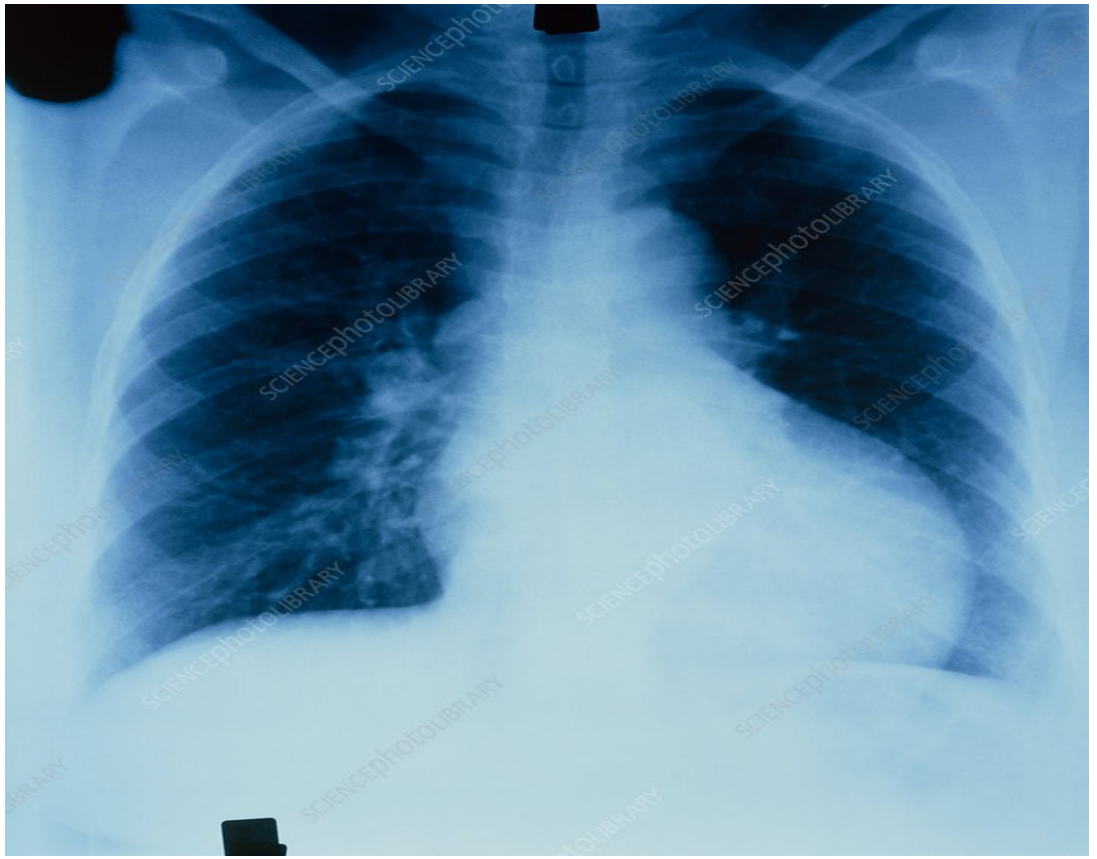
ECG showed left bundle branch block with a **wide QRS complex and left ventricular enlargement**



Easily fatigue with dyspnea on lying dow

Results of Medhat investigation:

Chest x-ray showed **cardiomegaly** with cephalization and **lung congestion**



Easily fatigue with dyspnea on lying dow

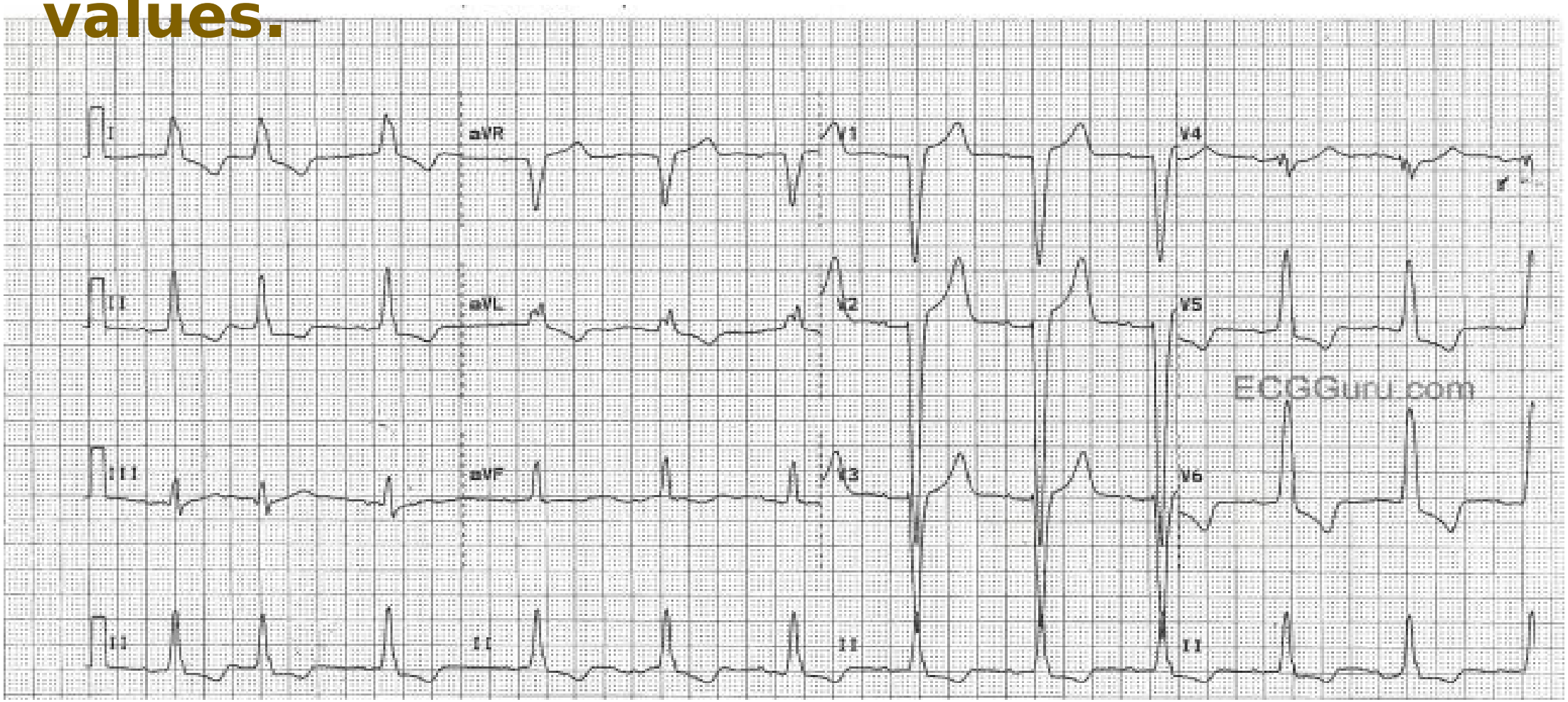
Results of Medhat investigation:

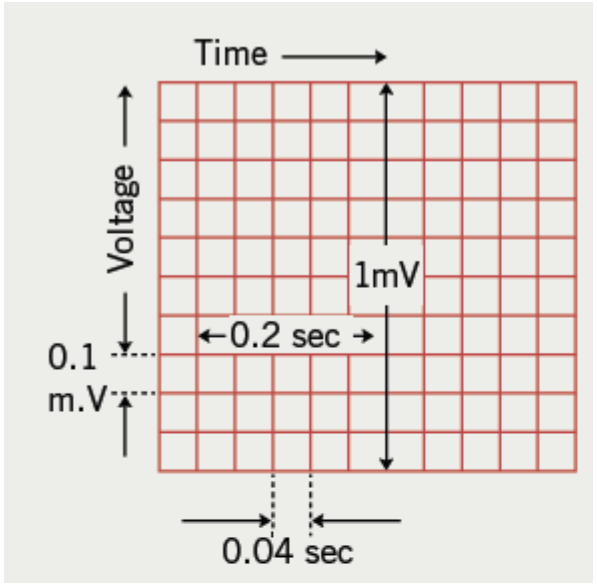
CBC, urea , creatinine were within normal

BNP was elevated

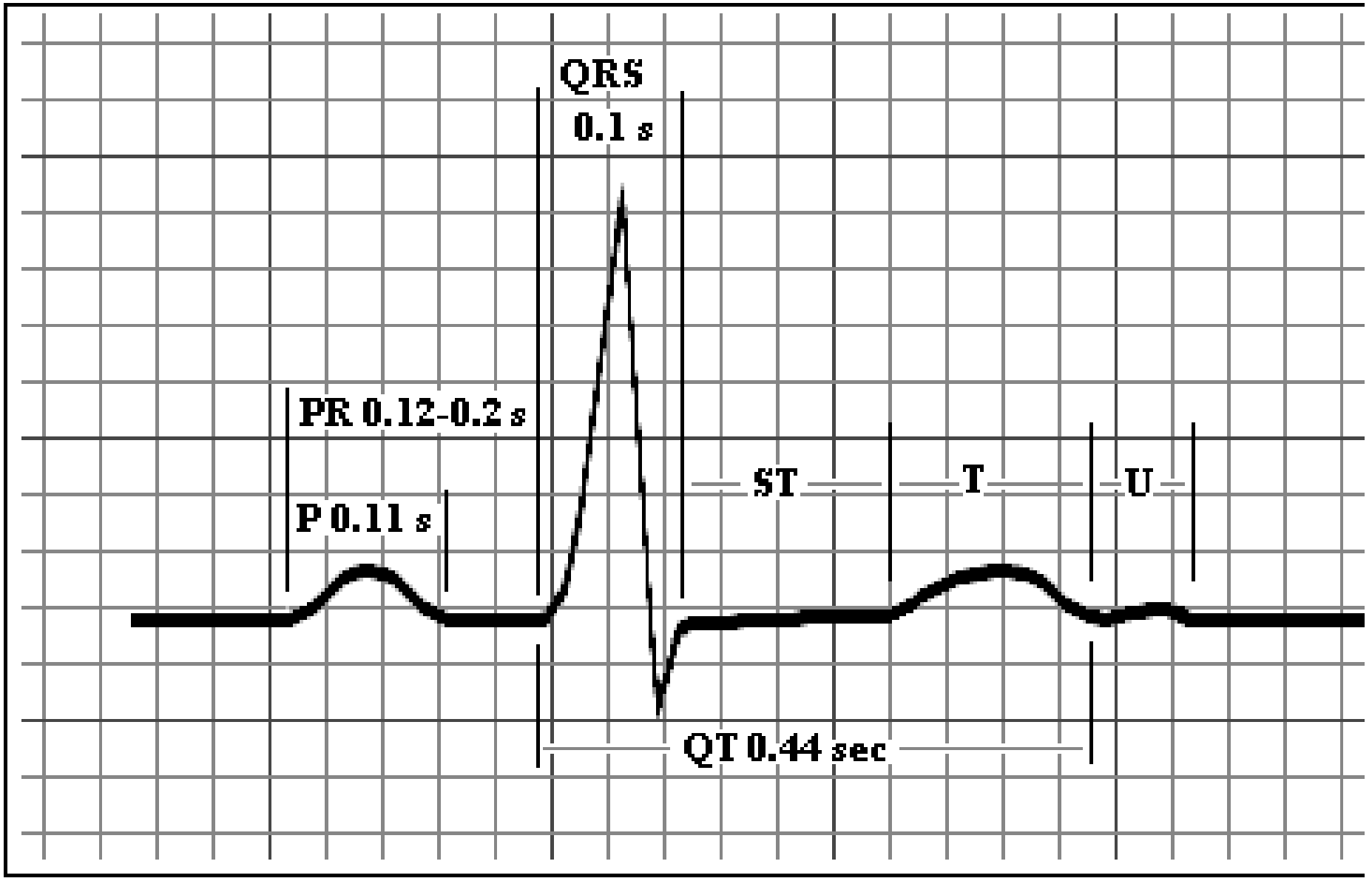
Sodium: 137 mEq/L
Potassium: 4.6 mEq/L
Liver functions showed: mild elevated bilirubin

6- What’s the duration of QRS complex in Medhat’s ECG? Compare this to normal values.





https://bjcardio.co.uk/files/uploads/2013/09/Figure-15.png

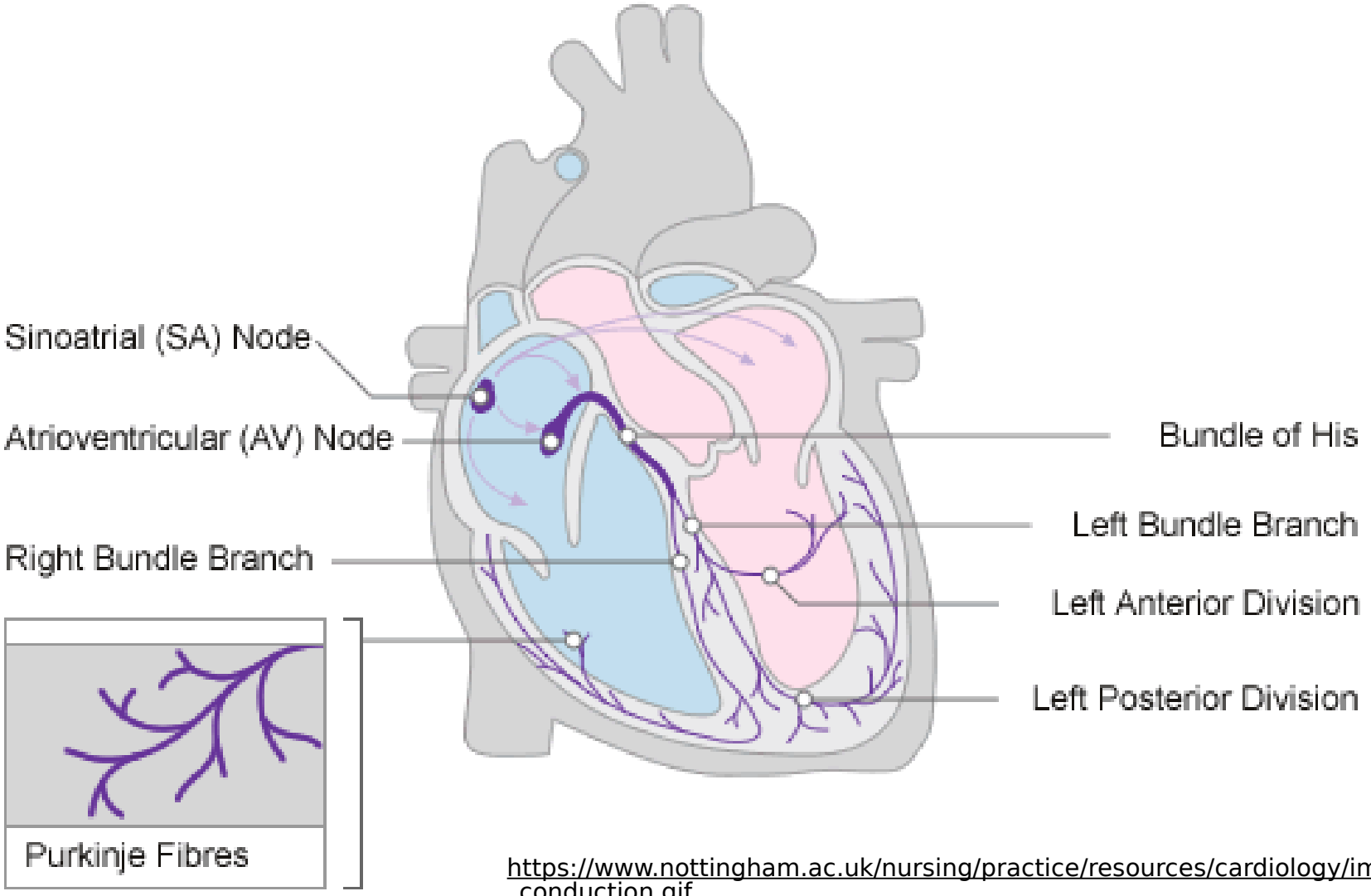


https://www.madsci.com/manu/ekg_pics/e_parts.gif

A wide QRS complex indicates slow conduction of impulse in the ventricular wall due to a defect in the conducting system, and this matches with the left BBB.

Describe the normal cardiac conducting system.

Cardiac conducting system



https://www.nottingham.ac.uk/nursing/practice/resources/cardiology/images/cardiac_conduction.gif

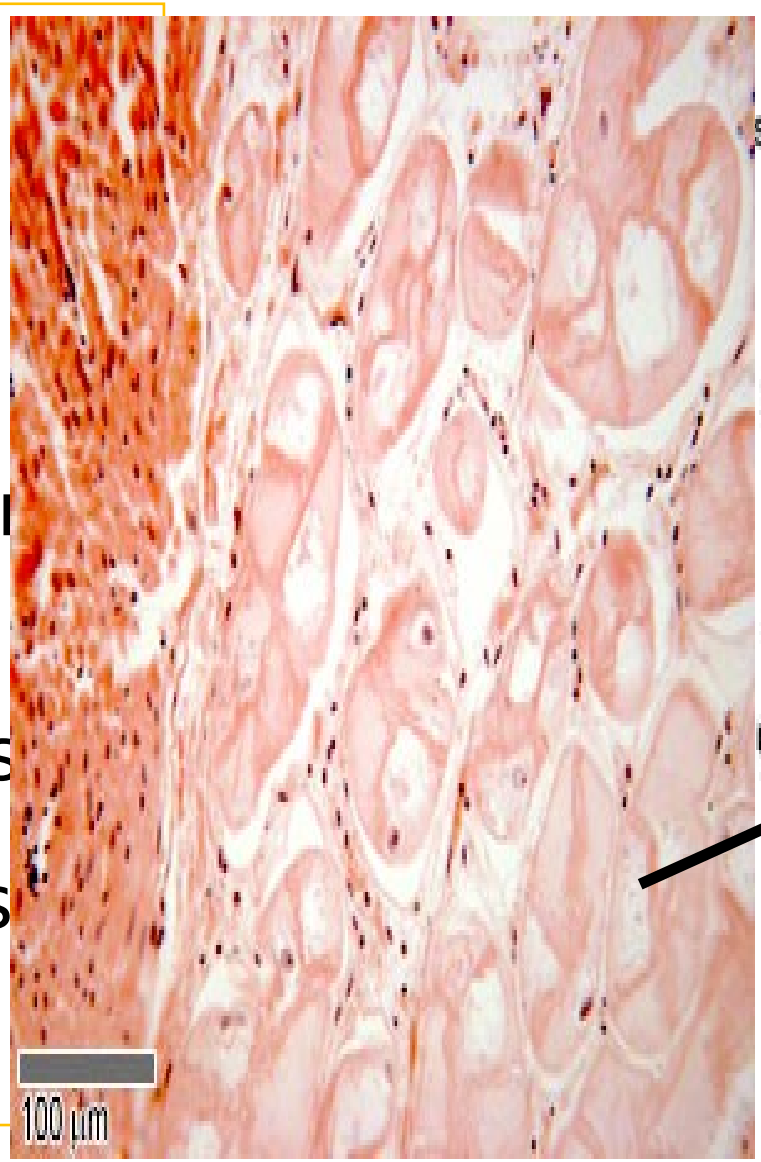
7- What is the histological structure of the conducting system of the heart?

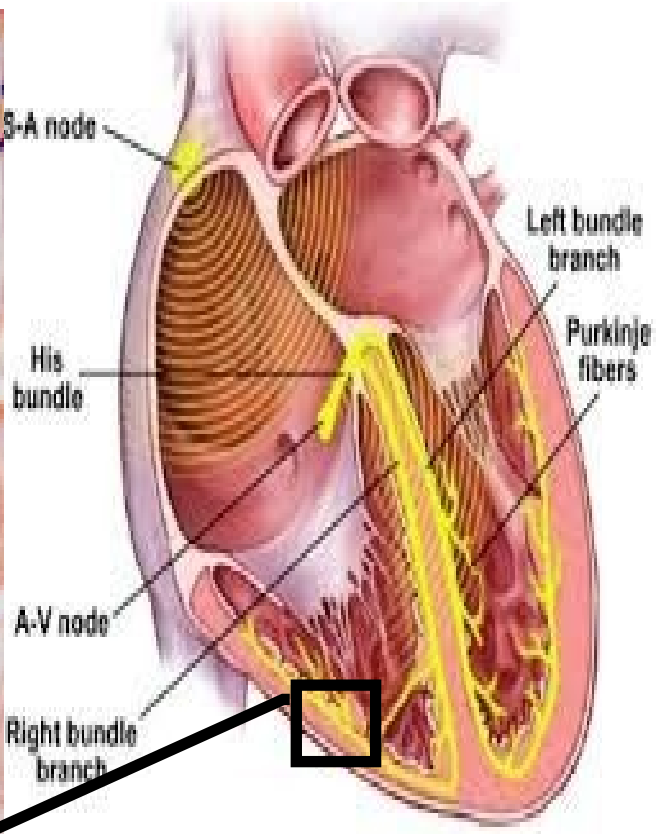
Conducting system of the heart

It is formed of modified cardiac muscle fibers called **Purkinje fibers** surrounded by C.T. sheath.

It is characterized by:-

- **Large** in diameter.
- **Pale & vacuolated** (rich i glycogen).....resist hypoxia
- **Few myofibrils**
- Richly supplied with nerves (autonomic).
- Rapid conduction of impuls
- No diad
- No tubular system.

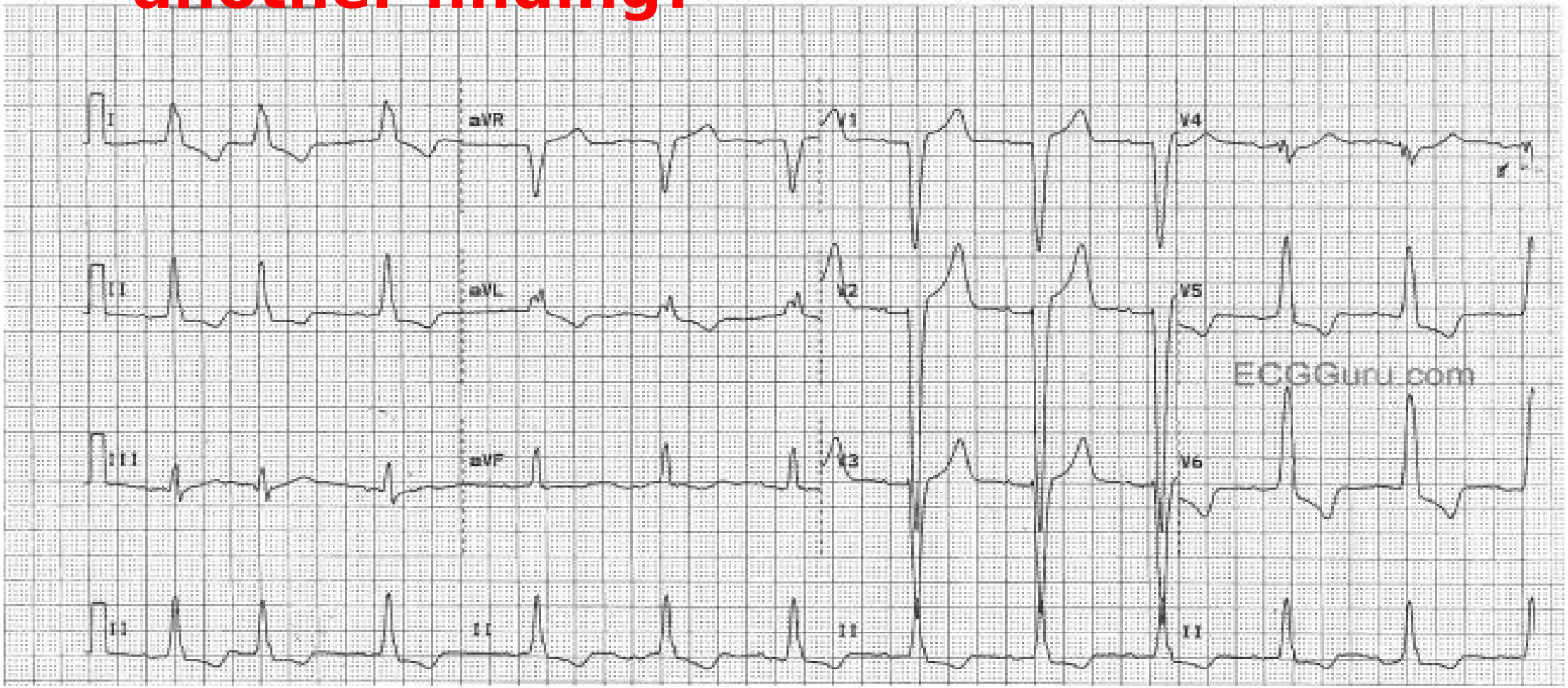




https://www.pinterest.com/pin/361413938820557426/?lp=true

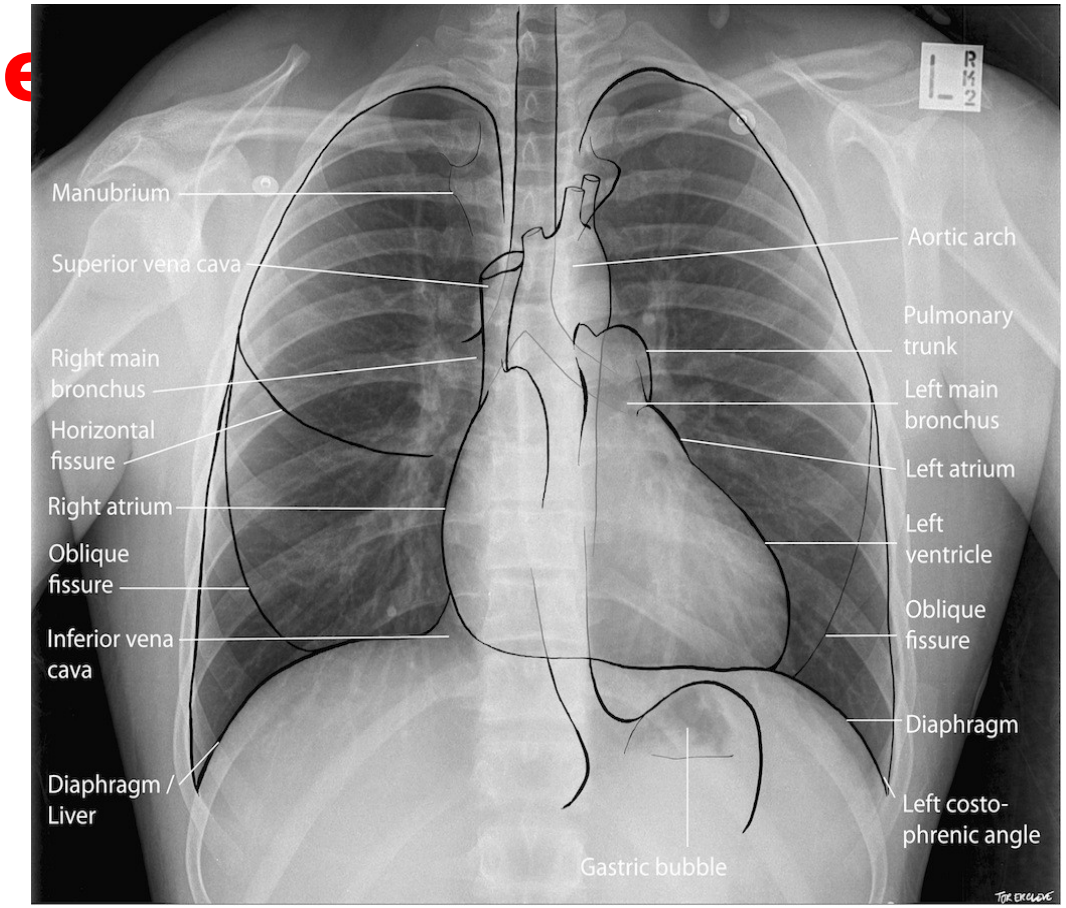
https://www.histology.leeds.ac.uk/circulator

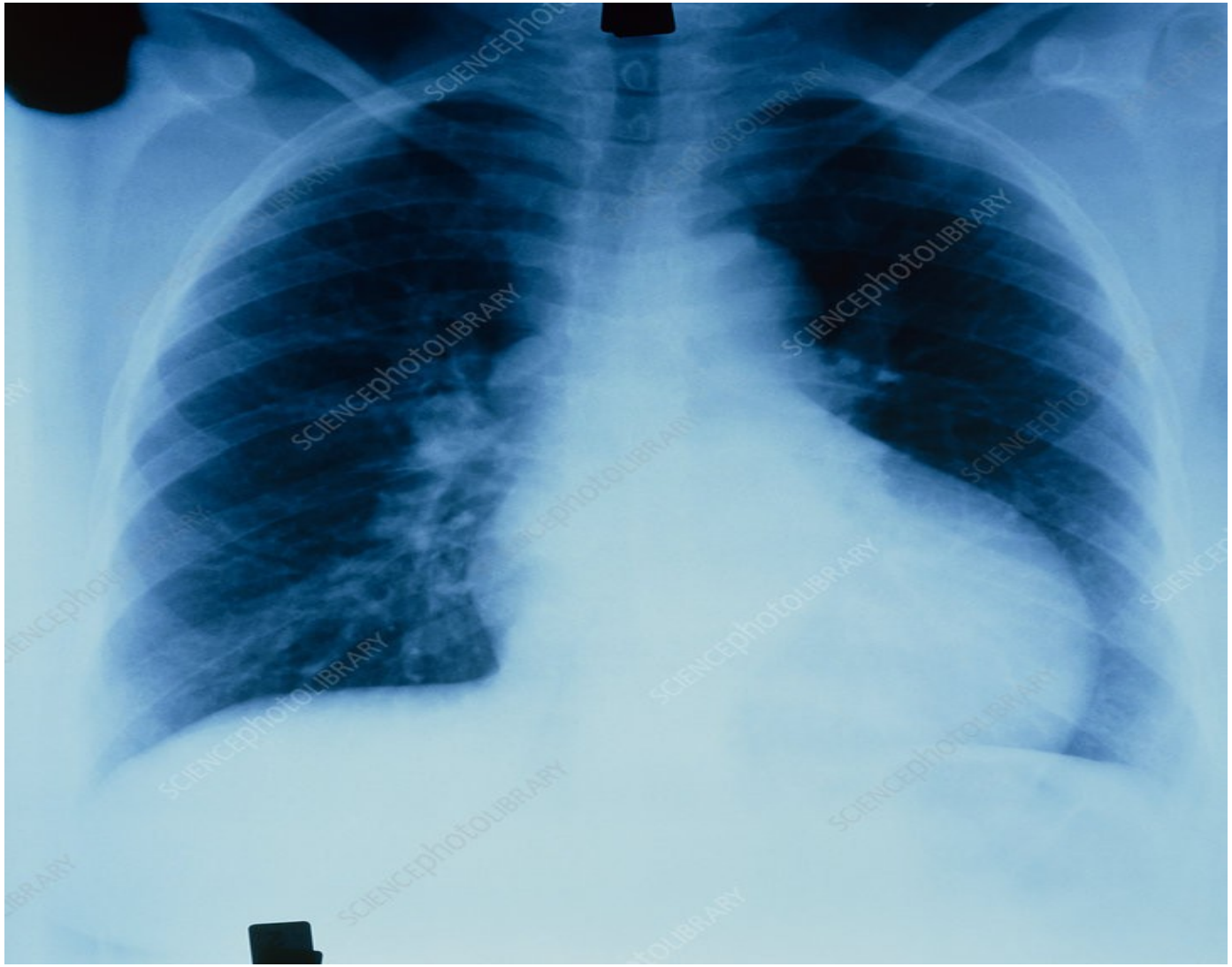
Interpret Medhat's ECG….Is there another finding?



8- Interpret Medhat's chest x-ray?

Which chamber is e



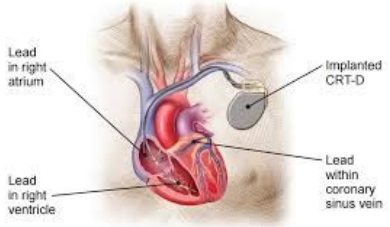


9- How could you manage Medhat?

- **Medical treatment**
  - **Diuretics** for symptom control
  - **ACEIs / ARNI**
  - **BBs** titrated to the maximum tolerated dose
  - **SGLT2 inhibitor**
  - **MRAs** titrated to the maximum tolerated dose

- **Lifestyle modification**

- **Consider digoxin if still symptor**



- **Consider CRT if with LBBB , QRS >130ms**
- **consider heart transplantation if all measures fail**

Welcome back

Easily fatigue with dyspnea on lying dow

Medhat was admitted to the ICU and given

- Oxygen supplementation
- IV 40mg **furosemide**
- **Ramipril** 10mg /day
- Rivaroxiban 20mg

After 24 hours, Medhat has symptomatically improved.

He stayed in the hospital for a week and carvidelol 6.25mg/day was added.

The doctor advised him to have low salt diet.

After discharge, patient was given **oral frusemide, Ramipril and Carviderol**

The doctor asked him to follow up after one week .

What is the mechanism of action of Furosemide?

Mechanism of action:

Loop diuretics **inhibit** the **cotransport of $Na^+/K^+/2Cl^-$** in the luminal membrane in **the ascending limb of the loop of Henle.**

-The loop diuretics are the most efficacious of the diuretic drugs.

-They releases renal PG→ ↑ renal blood flow

What are the adverse effects of Frusemide ?

Adverse effects

A. Hypovolemia

B. Hypokalemia

C. Hypocalcemia

D. Hypomagnesemia

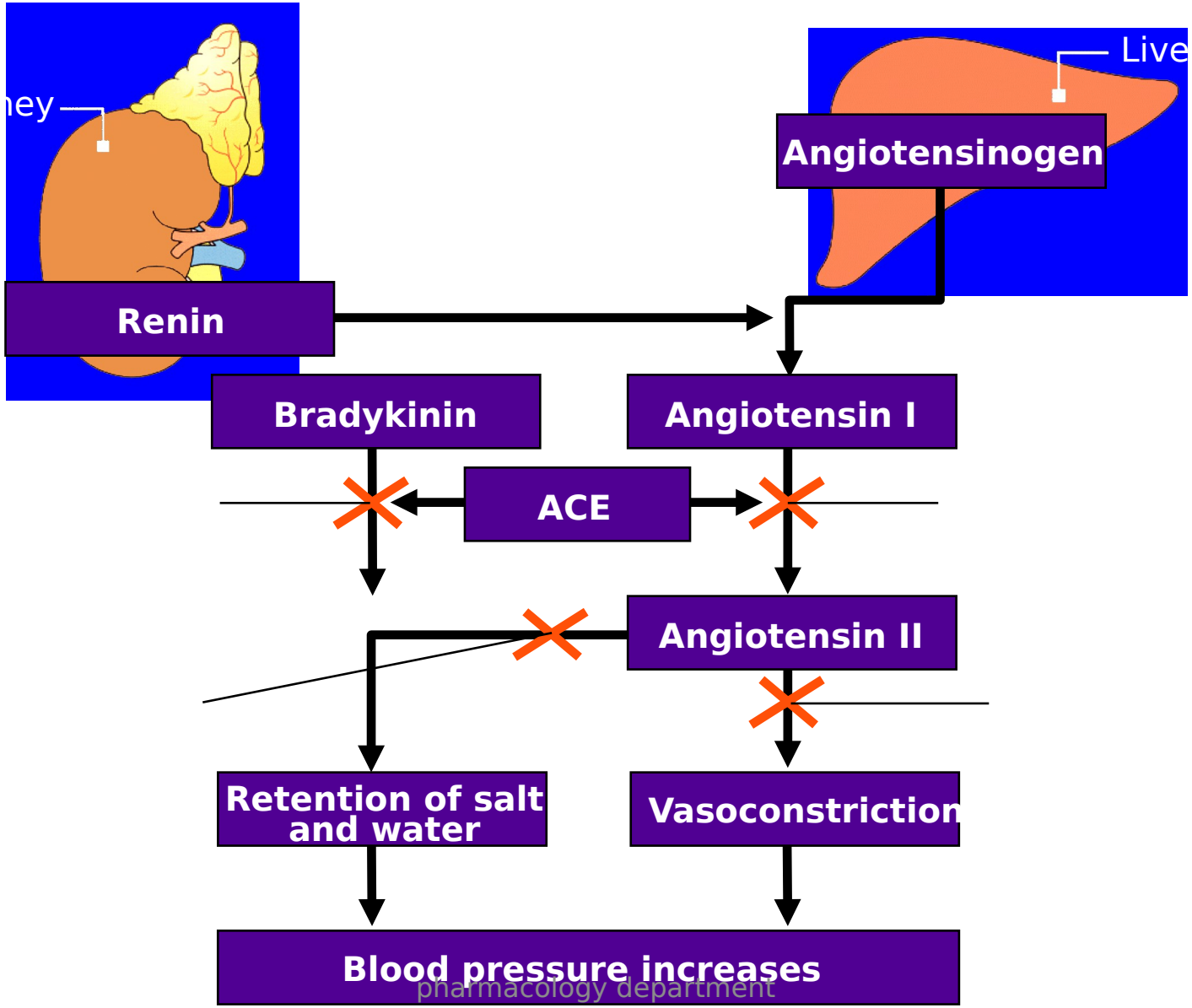
E. Hyperuricemia

F. Ototoxicity (with aminoglycosides)

G. Interstitial nephritis (with cephalosporines)

10- What is the mechanism of action of Ramipril ?

The renin-angiotensin-aldosterone system



Mechanism of action of Ramipril

- It is an ACE inhibitors with subsequent decrease in angiotensin II And increase in bradykinin vasodilation of arterioles and venules (so decreases both preload and afterload)
- Decreases NE release, vasodilatation without reflex tachycardia
- Decreases aldosterone release, decreases salt and water retention
- Decreases cardiac remodeling

What are the side effects of Ramipril?

MOST COMMON
Chronic dry cough

MOST SERIOUS
Angioedema

LESS COMMON

1-1st dose hypotension
2-Hyperkalemia:
3--
Hypersensitivity:
4- GIT:
- Anorexia- vomiting.
- Taste disturbance.

SERIOUS
1-Renal impairment (reversible):
More common in **high renin states**:
a. Patients on diuretics
b. Bilateral renal artery stenosis

2-Bone marrow depression.
3-Fetotoxic: if given in last trimester.
4-Teratogenic if given in 1st trimester.

What is the role of B-blockers (Carvedilol) in this case?

Role of beta blockers in HF:

1. ↓ HR → ↑ coronary flow.
2. ↓ Afterload and ↓ O_2 demand.
3. Cardioprotection: block catecholamine & Ag II - induced arrhythmia, myocardial damage & apoptosis.
4. Improve LV remodeling.
5. Resensitize the downregulated b_1 receptors (in long standing HF) → improve cardiac contractility.

What are the precautions should be followed in carvedilol therapy?

Carvedilol should be started at low doses and gradually titrated to effective doses.

Carvedilol should be used hemodynamically stable patients, after the use of ACEIs & diuretics

11- What is the role of ARNI in heart failure?

Angiotensin Receptor-Neprilysin Inhibitor (ARNi) is a medicine resulting from the combination of two anti-hypertensive drugs (sacubitril and valsartan) that reduce blood pressure.

The two active substances, sacubitril and valsartan, work in different ways. Valsartan is an angiotensin receptor blocker, while Sacubitril blocks the breakdown of natriuretic peptides produced in the body. Natriuretic peptides cause sodium and water to pass into the urine. This effect reduces the work on the heart and reduces blood pressure. The combined effect of the two medicines reduces the strain of the failing heart.

What is the role of SGLT2 inhibitors in heart failure?

Sodium glucose transporters 2 inhibitors are drugs used in treatment of type 2 DM,
Potential beneficial effects in heart failure include:
diuresis, and **reduction in blood pressure**.
More recently, novel mechanisms have been proposed to explain these benefits that include **improved cardiomyocyte calcium handling**.

Take Home Messages

-Chest pain must be adequately investigated and treated

- Paroxysmal nocturnal dyspnea strongly suggests cardiac cause of dyspnea

-Control of risk factors

-B Blockers in heart failure have many benefits.

Thank
you